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Agent Orange and the Prevalence of Cancer among the Vietnamese Population 30 Years after the End of the Vietnam War

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Abstract

During the Vietnam War, more than 70 million liters of military herbicide were sprayed over the combat zone. This study uses self and proxy-reported data on cancer status obtained from a nationally representative health survey of the Vietnamese population (N=158,019), combined with measures of military herbicide exposure computed from detailed information on US and allied wartime military activities. No significant difference in the prevalence of reported cancer is detected between communes with some degree of exposure and those with none. When restricting the analysis to exposed communes and adopting a continuous measure of herbicide exposure, there is evidence of a dose-response relationship; among communes that were exposed, increasing exposure to past military spraying is associated with increasing prevalence of reported cancer in 2001–2002. There is mixed evidence as to whether cohorts born before or after the end of the spraying campaigns are equally affected.

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Agent Orange and the prevalence of cancer among the Vietnamese population 30 years after the end of the Vietnam War[*]

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1. Introduction

During the Vietnam War (1959-1975), 72 million liters of herbicide were sprayed between 1961 and 1971 to defoliate forests and destroy food crops. Several herbicide mixtures were used, each named after the color of the stripe painted on the storage barrels. Agent Orange, the best known, accounts for 65 percent of the total (Stellman et al., 2003). Some mixtures (Agent Orange, Purple, Pink and Green) are characterized by a high concentration of 2,4,5-trichlorophenoxyacetic acid, contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (henceforth dioxin). An estimated total of 366 kilograms of pure dioxin was eventually sprayed, while the World Health Organization sets the tolerable daily intake to be in the range of 1 to 4 picograms (1 pg = 10^{-15} kg) per kilogram of body weight. Furthermore, the documented persistence of high levels of dioxin in Vietnamese food, soil and human tissues suggests that toxicity may be lingering well after the end of the war.[1]

However, establishing any health impacts of herbicide spraying is challenging. During the class action suit filed by the Vietnamese Association of Victims of Agent Orange/Dioxin against companies that supplied herbicides to the US military in Vietnam, senior district judge Jack Weinstein emphasized "*[t]he fact that diseases were experienced by some people after spraying does not suffice to prove general or specific causation*" (quoted in Stone, 2007). The Institute of Medicine publishes biennial updates on scientific knowledge about the effects of exposure to herbicides used during the Vietnam War (Institute of Medicine, 2007). A significant body of literature focused on US veterans. Unfortunately, the wealth of information used in US studies has no equivalent in Vietnam, a country with an estimated purchasing-power-parity GDP of $2,600 per capita in 2007, compared to $45,800 for the US (CIA, 2008).

This study explores whether, in the absence of detailed hospital records, a multipurpose household survey of the Vietnamese population (N=158,019) with self and proxy-reported information on individual morbidity can shed light on the health impacts of military herbicide spraying on civilians 30 years later. Combined with detailed information on US and allied military missions, the household dataset is used to analyze the relationship between measures

[1] Evidence of persisting high levels of dioxin in food is provided by Schecter et al. (2003, 2004), and Schecter, Malish and Ryan, (2003); for evidence of persisting dioxin in soil, see Schecter, Elitzer and Hiters (1989) and Tuan et al. (2007). Finally, Schecter et al. (1987, 1994, 1995, 1996, 2001, 2002, 2003, 2004, and 2006), the CDC (1988), Kahn et al. (1988), Quynh, Dai and Thom (1989) and Phuong et al. (1996) document the presence of dioxin in human tissues.

of past military herbicide exposure and the prevalence of (reported) cancer in the Vietnamese population some 30 years after the end of the war.[2]

When comparing communes that have been exposed with those that have not, no significant difference in reported cancer prevalence is detected. This finding could well reflect an attrition bias, since mortality due to cancer can induce a lower observed prevalence (stock), while still being consistent with a higher incidence rate (flow). A dose-response relationship is furthermore detected; conditionally on being exposed to past military spraying, increasing exposure is associated with increasing prevalence of self-reported cancer in 2001-2002. Although the findings could be driven by a causal impact of Agent Orange exposure on cancer, a careful interpretation of these results is called for, given the limitations of the data and methodology used for the analysis. First, information on cancer prevalence is obtained in a survey context and therefore subject to reporting biases; no validation has been undertaken, and there is no information on the types of cancer affecting individuals. Second, available data only allow computing prevalence rates rather than incidence rates potentially leading to attrition biases, although it might be reasonable to suppose that in this instance, coefficients would be biased towards zero. Third, the patterns of military herbicide spraying were surely not a random process, and responded to local conditions that could well be contemporaneous risk factors of cancer. Although a large number of confounders can be observed and controlled for, latent heterogeneity cannot be ruled out.

This paper belongs to the literature on the legacy of wars (for a review, see Blattman and Miguel, 2009). While the direct costs of wars in terms of human casualties and physical destruction cannot be over-emphasized, attention has also been given to the analysis of post-war economic recovery and long-term development. Organski and Kugler (1977) and more recently Chen, Loayza and Reynal-Querol (2008) find evidence of countries returning to pre-war economic development levels once peace has been restored. Evidence of long-term

[2] The US military, through the National Archives and Records Administration, has made available detailed information on wartime military operations. Kalyvas and Kocher (2009) analyze look at the determinants of violence using data from the Hamlet Evaluation System, a database of violence against civilians across hamlets in Vietnam. Closer to this paper in its methodology and data sources, Miguel and Roland (2006) instead use information on bombing activities to investigate their long-term impacts on economic development.

convergence is also supported in country-level studies; Davis and Weinstein (2002), Brakman, Garretsen and Schramm (2004) and Miguel and Roland (2006) document the catching-up of areas that were heavily affected by bombing in Japan, Germany and Vietnam, respectively. When health is the outcome of interest, war veterans have been the subject of most research efforts (almost all the peer-reviewed medical studies on the health effects of Agent Orange exposure is done on war veterans), while little attention has been paid to civilian populations; this might be partly due to lack of appropriate data. Exceptions include a cross-country analysis from Ghobarah, Huth and Russett (2003), which provides evidence that the indirect and lingering effects of wars significantly add to the burden of death and disability. At the micro-level, Alderman, Hoddinot and Kinsey (2004) and Akresh, Verwimp and Bundervoet (2009) look at the consequences of conflict on child malnutrition and long-term health, while Do and Iyer (2009) reviews the burgeoning literature on the effect of wars on mental health and provide an overview of methodological challenges that plague attempts to assess the impacts of violent conflict on mental health. The identification issues raised therein also apply in force to the present analysis.

The rest of the paper is organized as follows; section 2 provides background information on the Vietnam War of relevance to the present analysis. Section 3 describes the data used in the analysis and fleshes out the empirical methodology. Results are presented and discussed in section 4. Section 5 concludes.

2. The Vietnam War and the Agent Orange controversy

The Vietnam War or Second Indochina War took roots in the aftermath of the First Indochina War (1946-1954), which put an end to France's 70-year long colonial rule over Indochina and led to the partition of Vietnam into two, following the Geneva conference on July 21st, 1954.[3] A communist North Vietnam and a pro-West South Vietnam emerged on each side of the 17th parallel.

The start date of the war is commonly associated to the first guerilla attacks by North Vietnam against the South in 1959. Ho Chi Minh, North Vietnam's leader, had the objective to

[3] This section summarizes common facts known about the Vietnam War, and does not intend to account for all the aspects of the conflict. See e.g. Guilmartin (1991) for a more detailed outline of the wars in Vietnam since the French involvement.

reunite the country under communist rule. In December 1960, the government of Hanoi subsequently created the National Liberation Front (NLF) for South Vietnam. Initially, US involvement was restricted to providing technical and financial assistance to the government of South Vietnam in order to help the Army of the Republic of Vietnam (ARVN) combat the guerillas. Following the Gulf of Tonkin incident in August 1964 during which US ships were attacked off the Vietnamese coast, the US stepped up its military presence; the first American troops arrived in Vietnam in 1965. After enduring severe defeats, the North Vietnamese and the NLF launched the so-called Têt offensive in 1968 against multiple cities in South Vietnam. Although the communist forces lost on the ground and suffered heavy casualties, the political and psychological repercussions were significant; President Lyndon Johnson did not seek reelection and Richard Nixon came into office with the plan of ending US involvement while building up the ARVN. On January 27th 1974, the Paris peace accords ended the conflict; all American troops had then left Vietnam. Soon after, North Vietnam resumed hostilities and rapidly dominated the ARVN. The communist forces entered Saigon, capital of South Vietnam, on April 30, 1975. The Vietnam War has ended and reunification of the country under communist rule ensued.

One controversial feature of Vietnam-era military warfare is the extensive use of military herbicides. Over the 1961-1971 period, an estimated 72 million liters were sprayed by US and allied forces, with the objective of depriving the enemy of cover and food.[4] Some herbicide mixtures were contaminated with dioxin; an estimated total of 366 kilograms of pure dioxin was eventually sprayed (Stellman et al., 2003). Table 1 is borrowed from Stellman et al. (2003) and provides a breakdown of herbicide mixtures used during the war. Each mixture got its common name from the stripe painted on the storage barrels. Agent Orange was the most used, accounting for 65 percent of total herbicide quantities used.

[TABLE 1 ABOUT HERE]

As early as 1977, reports of health symptoms affecting Vietnam War veterans triggered the US government to order studies to assess the possible causal link between herbicide exposure and health effects observed among veterans. The US Congress passed the 1991 Agent Orange

[4] Most (but not all) of herbicide spraying missions were part of Operation Ranch Hand, the code name for the spraying of herbicides by the US Air Force from 1962 to 1971.

Act, requesting the National Academy of Sciences to assess the health consequences of exposure to wartime military herbicides. The Institute of Medicine provides updates on the scientific evidence on a bi-annual basis.

The debate has also moved to the legal battlefield. War veterans and Vietnamese civilians filed a lawsuit against the chemical companies that manufactured the herbicides. The case was dismissed on March 10, 2005 and went to The Supreme Court, which let the dismissal stand in a March 2, 2009 decision.

3. Data and empirical methodology

3.1. Data

To look at the effects of herbicide spraying on cancer status in the Vietnamese population, two important sets of data are combined.

3.1.1. The 2001-2002 Vietnam National Health Survey

Self-reported and *proxy-reported* cancer status is collected from the 2001-2002 Vietnam National Health Survey (VNHS). VNHS is a nationally representative survey undertaken by the Ministry of Health and the General Statistics Office. A stratified random sample of 1200 communes (out of a total of 8926 communes) and 36 000 households was interviewed from November 2001 to November 2002. VNHS gathers individual data on self and proxy-reported morbidity. More precisely, the survey instrument asks the main respondent (in most cases the household head or his/her spouse) about all household members' health conditions. In particular, information on cancer is recorded as follows (official English translation): "May I ask, has anyone in your household been diagnosed by a doctor or PA with any of the following illnesses? 1) Blood pressure disease?, 2) Cancer?, 3) Epilepsy?". A positive answer is marked with an X in the column corresponding to a given health condition and in the line corresponding to the individual suffering from it. The outcome variable of interest in this study is derived from answers to the second question.

Socio-economic status is measured using elementary assets, education and food consumption information. Another survey module gathers information on individuals' health care utilization history. Finally, households' communes of residence are matched with the official General Statistics Office list of Vietnamese communes, which provides geographical coordinates of commune administrative centers.

3.1.2. Herbicide Report System

In addition to the VNHS, measures of military herbicide exposure are constructed using the US Military Assistance Command Data Management Agency's Herbicide Report System (HERBS) file. The data include but are not restricted to military operations conducted under Operation Ranch Hand. Both aerial (fixed wing, helicopter) and ground (truck, backpack) missions are recorded. A geographic information system (GIS) was further developed to facilitate the estimation of exposure from HERBS file data (Stellman et al., 2003; Stellman and Stellam, 1986, 2003, 2005). The GIS was initially designed to investigate the effect of Agent Orange and other herbicides on US veterans. The position of the majority of US military units is known with relative precision. These include largely those units that moved rarely or not at all, such as those assigned to fixed bases; locations are also known for many mobile units. For the Vietnamese population however, the issue is different; the current position of the household, let alone its position at the time spraying took place, is not accurately known so that exposure opportunity indexes computed by Stellman et al. (2003) and Stellman and Stellman (1986, 2003, 2005) cannot be used in the context of this investigation.

The GIS, nonetheless, allows counting the number of instances an herbicide spray application fell within .5, 1, 2 and 5 kilometers of a given reference point, and can also restrict counting to dioxin-contaminated herbicide applications only. However, by design, the buffer zone cannot be arbitrarily set by the GIS user. As a commune in Vietnam has a radius of typically 5 kilometers, the available measures become problematic: most households would live at the border of such 5-kilometer buffer zone, while herbicides have most likely been sprayed over rice fields and forests, further away from administrative commune centers. To overcome this shortcoming, buffer zones are extended by aggregating hit counts over a set of seven juxtaposed 5-kilometer-radius discs. The first disc - central disc - is centered on a commune center. The six other discs - peripheral discs - are centered on six points placed at a 10-kilometer distance, forming a regular 20-kilometer-diagonal hexagon as illustrated in figure 1. For consistency, the six peripheral points are chosen so that the hexagon has one North-South diagonal at constant longitude. Aggregating hit counts over these seven discs gives an approximate account of the number of spray applications that occurred within 15 kilometers of a given reference location. This measure, the *herbicide hit count* measure, is the main proxy measure of exposure used in the study. Alternatively, the *dioxin-only hit count* restricts itself to dioxin-contaminated spray applications (Agent Orange, Purple, Pink and Green).

[FIGURE 1 ABOUT HERE]

3.1.3. Combat activity files and bombing intensity information

To control for other potential determinants of conflict intensity, further data on combat activities provided by the Information Management and Mine Action Programs (iMMAP) are included in the analysis. Records combine the Combat Activities file (CACTA), the South-East Asia Database (SEADAB) and the Combat Naval Gunfire File (CONGA), and cover combat activities of allied forces over the period October 1965 - June 1975 with the exception of few missing months. The dataset contains the following fields: aircraft type, ordnance type, quantity, load, and drop date and location. Drop location is recorded when the drop actually takes place, which might differ from impact location. For example, in the case of B52 carpet bombing, the drop location of general purpose bombs refers to the north-west corner of the 1.5-kilometer*1.5-kilometer square, which was considered the most accurately recorded point of the box (for further details, see Miguel and Roland, 2006).

To be consistent with the herbicide exposure index, a measure of bombing intensity is constructed by aggregating bomb load across all categories and over a 15-kilometer buffer zone around commune centers. A binary variable indicating whether a commune has been bombed at all within 15km of its center and the logarithm of aggregate bomb load will henceforth be the adopted measures of bombing intensity.

3.1.4. Other data sources

Additional information on agricultural herbicide use, cigarette and alcohol consumption, and coal and firewood expenditures are obtained from the 2004 Vietnam Household Living Standards Survey (VHLSS) to look at household-level confounding factors. The General Statistics Office undertook a nationally representative Vietnam Household Living Standards Survey (VHLSS) in 2004. A total of 9000 households in 3300 communes were administered a detailed food and non-food expenditure survey module. From this dataset, herbicide and pesticide expenditures per area of agricultural land are computed, and averaged by surveyed communes. Similarly, household per capita expenditures in coal and firewood, cigarettes and alcohol are computed and averaged by communes. Exposure measures are assigned to each VHLSS commune to look at the cross-sectional association between past military herbicide exposure and current agricultural herbicide/pesticide, coal, firewood, cigarette and alcohol expenditures. Unfortunately, the overlap between VHLSS and VNHS communes is not significant enough to combine these two datasets, and explicitly control for these variables in the regressions.

To control for environmental confounders, this dataset is further combined with data on daily sunshine information for the period 1975-1999. Sunshine data were provided by the Meteorological Data Center (MDC), an institution under the Ministry of Natural Resources and Environment of Vietnam. The data were collected on a daily basis for the period 1975-2006 from 108 weather stations spread across the country. Each commune in VNHS was assigned the closest weather station and hours of daily sunshine were averaged over the period 1975-1999.

3.2. Empirical methodology

To look at the determinants of reporting cancer, we estimate the following multivariate logistic model:

$$ln\frac{p_{ijk}}{1-p_{ijk}} = \alpha + \beta\ Herb_k + X_{ijk}\gamma + \varepsilon_{ijk}$$

where p_{ijk} is the probability that individual i in household j living in commune k reports suffering from some form of cancer in 2001-2002, $Herb_k$ is the measure of wartime herbicide exposure for commune k, and X_{ijk} is a vector of individual demographic and socio-economic characteristics such as age (and age squared) and schooling, household-level socio-economic status (per capita consumption), and community-level location (urban or rural area), sunlight exposure (commune-level 1975-1999 average daily hours of sunshine) and wartime bombing intensity (bombing load); ε_{ijk} is the error term that is assumed to be potentially characterized by heteroskedasticity and spatial auto-correlation. Robust standard errors are computed accordingly, clustered at the provincial level in order to account for potential spatial correlation.[5] Estimations are moreover weighted using population weights provided in the dataset to account for study design.

Effects of exposure for cohorts born respectively before and after the end of herbicide application campaigns are estimated jointly by adding interaction terms (exposure index

[5] As of 1999, Vietnam was divided into 8926 communes, grouped into 61 provinces.

interacted with a binary variable indicating whether an individual is born after 1971) in the specifications:

$$ln \frac{p_{ijk}}{1-p_{ijk}} = \alpha + \beta\ Herb_k + \delta\ Herb_k \times 1_{Birth\ Year_{ijk}>1971} + X_{ijk}\gamma + \varepsilon_{ijk}$$

The coefficient δ will indicate whether or not exposure affected cohorts born after the end of the herbicide spraying campaigns differently from older cohorts that were potentially directly exposed.

4. Results and discussion

This section documents the associations between the prevalence of (self-reported) cancer in a Vietnamese commune in 2001-2002, and the extent to which that same commune was sprayed with military herbicides in the past. Table 2 describes the variables used in the analysis and summarizes the distribution of herbicide spraying activities. Almost half of the survey communes (593 out of 1200) have been sprayed at least once within 15 kilometers of the commune center; this figures decreases slightly to 568 if hit counts are restricted to dioxin-contaminated herbicides only. Note that the overall prevalence of cancer is 0.08 percent.

[TABLE 2 ABOUT HERE]

4.1. Herbicide exposure and cancer prevalence

Table 3 displays the results of multivariate logistic regressions of the determinants of cancer. The number of observations drops from 158,019 (columns 1, 4, and 7) down to 148,022 when education and consumption are controlled for (columns 2, 5, and 8), due to missing information in the survey. In columns 3, 6 and 9, an additional variable indicating whether the individual has been seeking either inpatient or outpatient care in the past year is added to the regression specifications.

Columns 1 to 3 look at the socio-economic determinants of cancer. Prevalence of cancer is higher among women, and the odds of cancer are increasing and concave with respect to age. Living in an urban area is associated with higher odds of reporting cancer. Wartime bombing intensity does not show any correlation with the prevalence of cancer, and neither does sunlight exposure. On the other hand, there is a strong association between reporting cancer and health care utilization.

Columns 4 to 9 document the association (or absence thereof) between the odds of cancer and two measures of herbicide exposure. These two measures are binary variables that take value 1 if the individual lives in a commune that experienced either (i) at least one herbicide spray application (columns 4 to 6), or (ii) at least 39 herbicide spray applications (columns 7 to 9), respectively, and 0 otherwise.[6] The results do not support any difference in cancer prevalence between a commune previously exposed to military herbicides and a commune never exposed, where exposure is defined by either one of the two aforementioned measures.

[TABLE 3 ABOUT HERE]

Table 4 undertakes a dose-response analysis. Instead of comparing communes with and without history of exposure, the analysis focuses on these communes that have had at least one herbicide spray application by the end of the war; a comparison of cancer prevalence is hence made across communes that have all been exposed to military herbicides, but at varying intensity levels. In this instance, a continuous exposure measure – the logarithm of herbicide hit count – is adopted. In columns 1 to 6, the continuous measure of exposure is added to the specifications used for table 3. On the one hand, communes with one herbicide hit count (i.e. for which the logarithm of herbicide hit count is equal to zero) exhibit *lower* reported cancer prevalence than communes with zero herbicide hit count. On the other hand, a dose-response relationship is detected. The results suggest that among communes that were previously exposed, an increase of 10 percent in herbicide exposure is associated with a 2 percent increased probability of reporting suffering from cancer. Equivalently, an individual currently living in a commune at the 75th percentile of the exposure distribution (91 hits) is estimated to have almost twice the risk of cancer than an individual with similar characteristics living in a commune at the 25th percentile of the exposure distribution (18 hits).[7]

By adding an interaction term with a binary variable indicating whether the individual was born after 1971, the end of herbicide spraying campaigns, columns 4 to 6 find that the dose-response relationship is weaker for post-1971 cohorts and no longer statistically significant at conventional confidence levels. Instead of looking at the full sample, the regressions presented

[6] A commune that experienced at least 39 herbicide spray applications is among the top 25-percent most sprayed communes.

[7] For the dose-response analysis, the exposure distribution considers only the universe of survey communes that experienced at least one herbicide spray application by the end of the war.

in columns 7 to 12 drop observations from individuals living in communes with no record of past herbicide spraying. The results are comparable to the full-sample analysis; cancer prevalence is positively correlated with the adopted continuous measure of herbicide exposure. In this case, however, no significant difference is found between pre and post 1971 cohorts and the effect of exposure on post-1971 can be detected at a 10 percent confidence-level , except when health care utilization is controlled for (column 12).

[TABLE 4 ABOUT HERE]

Tables A1 and A2 in the appendix perform the same analyses as in tables 3 and 4, restricting to *dioxin-only* spray applications to construct measures of exposure. Although the results look similar, they are not statistically significant at conventional confidence levels.

4.2. Discussion

A discussion of the results – the existence of a positive dose-response association between past herbicide exposure and contemporaneous prevalence of cancer, despite a documented attrition bias – attempts to assess the limitations of the adopted approach, and to debate a causal interpretation of the estimated correlations. Three issues need to be addressed: (i) the use of self-reported cancer status to construct prevalence measures, (ii) the adopted measure of herbicide exposure, and (iii) potential omitted variable biases.

4.2.1. Self reported cancer status

An overwhelming majority of studies that aimed at investigating a potential link between dioxin and cancer were performed on non-Vietnamese war veterans, for whom hospital records are available or biopsies and X-ray screenings were performed (with few exceptions that also relied on self-reports; see e.g. Kang et al., 2006). With regards to Vietnam, such information is not systematically available, and national cancer registries are subject to the caveat that surveillance might be positively correlated with herbicide spraying; the government might have encouraged cancer screening in areas more heavily sprayed, which would then be a source of bias in any attempt to correlate cancer prevalence obtained from registries and the intensity of herbicide spraying.

An alternative is to use a large-scale survey with the understanding that the analysis needs to rely on self-reported or proxy-reported health outcomes. In this study, responses for each household member are reported by the survey respondent who typically is the household head or his/her spouse. No validation of the survey instrument has been done. In addition, the study does not allow for precise assessment of the type of cancer an individual is suffering from. Although a study executed in the US by Dominguez et al. (2007) found self-reported cancer history to have 85.7 percent sensitivity (ranging from 92.1 percent for breast cancer to 42.9 percent for leukemia), no comparable exercise is known to have been conducted in a developing country setting. Although the approach adopted in this study has its limitations, it is believed to have an added value given the scarcity of alternative approaches (Phuong et al., 1996; Kamarova et al., 1998).[8] To address the issue given current data availability, few variables are aimed at addressing heterogeneity in accuracy of self-reports: individual's education and expenditure levels, an urban/rural binary variable and a binary variable indicating whether inpatient or outpatient care has been used in the past 12 months. These variables partially capture awareness about health status or access to screening facilities, as well as other risk factors (such as pollution in urban areas), occupational risk (correlated with income or education), or reverse causation (cancer-affected individuals are more likely to seek care). Yet, controlling for these variables barely affects the observed association between herbicide exposure and cancer prevalence.

Nevertheless, it might well be the case that individuals who are aware that their communes of residence were heavily sprayed in the past are more likely to report suffering from cancer, leading to recall biases.[9] Meanwhile, evidence presented in table 3 does not show any difference between a commune with zero hit count and a commune with a strictly positive hit count (columns 4 to 6). Neither does it show any difference between communes below and above the 75th percentile cutoff (columns 7 to 9). If biased reporting was driving the observed correlation between cancer prevalence and herbicide exposure, one would expect to detect an association with a binary measure of exposure (table 3) rather than a continuous one (table 4), since individuals should remember whether or not they were exposed before remembering how heavily they were exposed. In addition, the estimations shown in table 4 (columns 1 to 6)

[8] If self-reported cancer status was totally uninformative about true cancer status, then no association would be detected. Results presented in table 4 suggest otherwise. Random measurement errors of the dependent variable inflate standard errors, without biasing the regression coefficients.

[9] Note that if the use of hospital records was available, the same type of bias would still prevail: individuals who believe they have been sprayed would be more likely to get screened for cancer.

suggest that if anything, a commune with no exposure reports *fewer* instances of cancer than a commune that experienced one herbicide spray application.

4.2.2. Measures of herbicide exposure

Unlike in earlier studies, levels of dioxin actually absorbed by each surveyed individual are unknown. Instead, information about geographic location of herbicide spray applications is used to construct the exposure measures. The use of military records or self-reported exposure assessments as measures of exposure in the case of war veterans has been subject to debate (Young, 2004; Young, Cecil and Guilmartin, 2004).[10] Furthermore, some studies based on military records draw conclusions of non-occupational exposure (Michalek and Pavuk, 2008; Pavuk, Michalek and Ketchum, 2006; O'Brien, Decoufle and Boyle, 1991). In the context of the present analysis, past exposure at the current commune of residence is the adopted measure of herbicide exposure. Evidence of some positive correlation between measures constructed from military records and actual dioxin levels in adipose tissue in residents of South Vietnam has been documented by Verger et al. (1994). This could be evidence of direct wartime exposure, or exposure via dioxin-contaminated food, soil or river sediments.

A first concern is that military record data might not reflect true exposure on the ground. Dioxin-contaminated river sediments and fish might be located in places further downstream from herbicide application areas, while geographical conditions and soil movements have been found to affect the spatial distribution of dioxin contamination (Phuong et al., 1996). In addition, around 8 percent of recorded missions did not refer to a particular herbicide mixture although Agent Orange was most likely used (Stellman and Stellman, 2005). The lack of robustness of the results to the use of measures of exposure derived from dioxin-contaminated herbicides might be attributed to such increased measurement error (appendix tables A1 and A2). Finally, the construction of the exposure measure itself is a source of measurement errors. The number of hits does not convey information on either the actual chemical composition of a spray application or the quantity sprayed. Moreover, the exposure opportunity measure is computed by aggregating over space, without taking into account spatial or time decay. However, as measurement errors are believed not to be correlated with cancer prevalence,

[10] Michalek and Pavuk (2008), Pavuk, Michalek and Ketchum (2006), Pavuk et al. (2005), Bullman, Watanabe and Kang (1994), Mahan et al. (1997), Dalager et al.(1991, 1995), Kang et al. (1986, 1987) and Anderson (1990) use military records to construct proxy measures of exposure opportunity; Giri et al. (2004) and Zafar and Terris (2001) rely instead on self-assessed exposure.

estimates are biased towards zero, providing a conservative assessment of the true association. It is for example unlikely that areas where records were incomplete are also areas with lower prevalence of cancer; soil movements, while affecting the measurement of exposure, are otherwise not believed to have an effect on the prevalence of cancer. Such “classical” measurement errors in the right-hand side variable are therefore expected to an attenuation bias; the results are biased towards *not* finding an association between exposure and the prevalence of cancer.

Second, individual-level data used to conduct the analysis are taken from a cross-sectional survey done 26 years after the end of the Vietnam War, with no retrospective information. Thus, two additional issues might arise: one relates to a change of location of individuals between spray applications and present time (e.g. soldiers in sprayed areas returning home at the end of the war or households recently settled in the survey commune), and the other to the fact that the household survey captures information on cancer only for people alive at the time of the enumeration. Population movements or “natural” attrition due to death unrelated to dioxin contamination lead to an increased measurement error in the outcome variable, which adversely affects the precision of the estimation without biasing it.[11] Finally, if higher exposure is believed to increase mortality rates, then areas more heavily sprayed will exhibit a *lower* prevalence of cancer; the negative coefficients on the binary exposure measure in table 4, columns 1 to 6, are consistent with such potential attrition bias.

4.2.3. Omitted variables and causal interpretation of the findings

Other potential cancer risk factors can confound the positive association between exposure and cancer observed in table 4.

Herbicide spray application campaigns are not a random process, but responded to local conditions that may persist until present times and be factors of cancer. Alternatively, bombing and spraying campaigns can in turn create local conditions that are themselves risk factors for cancer. Variables measuring past bombing intensity are added to all the regression specifications in order to capture some of the common geographical determinants of both bombing and herbicide spraying campaigns (the correlation between bombing and spraying is

[11] The effect of soldiers returning home from heavily sprayed areas would on the other hand tend to bias results towards not finding any association between exposure and cancer prevalence. Furthermore, administrative restrictions severely reduced the scope for migration. VNHS however does not contain migration information to explicitly investigate the issue.

0.32, p<0.01). To address other potential confounders, sunlight exposure is controlled for, by using a variable measuring commune average daily sunshine over the period 1975-1999. Socio-economic status, measured using education levels and per capita expenditure, and an urban/rural binary variable are also added as control variables in the regression specifications.

In addition, it is plausible that areas more heavily sprayed in the past were areas of dense forest, or more generally agro-climatic zones that require more herbicide (and pesticide) use to maintain high agricultural productivity. Thus, cancer outcomes might rather be driven by contamination due to agricultural herbicides and pesticides. Contemporaneous measures of herbicide and pesticide use, however, show a negative correlation (correlation = -0.1082, p<0.01) with past military herbicide application intensity (figure 2, upper left panel). Finally, herbicide spraying might also be correlated with other risk factors of cancer: smoking, alcohol, and occupational risk such as the use of coal and firewood for energy consumption. Figure 2 also displays the correlations between the patterns of herbicide exposure and commune-level averages of household per capita expenditure of coal and firewood (upper right panel), cigarettes (lower left panel) and alcohol (lower right panel). If anything, the correlations appear to be negative.

[FIGURE 2 ABOUT HERE]

5. Conclusion

This study quantitatively assessed the link between herbicide exposure and the prevalence of cancer in the Vietnamese population 30 years after the end of the Vietnam War. Since systematically replicating methodologies that have been conducted among US veterans is beyond Vietnam's current capacity, an alternative approach was adopted instead. Using self- and proxy-reported measures of cancer prevalence, a positive association between past exposure to military herbicides and current prevalence of cancer is detected in the data. Admittedly, the adopted methodology is constrained by the reliability of self- and proxy-reported cancer status, the use of prevalence rather than incidence as outcome measures and the lack of further information on types of cancer. Until further studies are undertaken, there is however a risk of losing the evidence with the disappearance of most affected older cohorts.

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Table A2: Logistic regression of the determinants of cancer - dose response to dioxin-only hit counts

	Dependent variable: Individual reports suffering from cancer (1:yes,0:no)											
	Full sample						Conditional sample					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Independent variable												
Logarithm of dioxin-only hit count	1.131	1.134	1.145	1.164	1.171	1.184	1.162	1.170	1.196	1.160	1.168	1.198
	(0.127)	(0.130)	(0.135)	(0.132)	(0.137)	(0.143)	(0.144)	(0.150)	(0.156)	(0.134)	(0.140)	(0.146)
Additional effect of log dioxin-only hit count on post-1971 cohorts				0.760	0.751	0.750				1.022	1.015	0.979
				(0.153)	(0.154)	(0.152)				(0.332)	(0.336)	(0.326)
Individual is born after 1971 (1:yes,0:no)				1.167	1.251	1.271				1.075	1.389	1.416
				(0.626)	(0.676)	(0.670)				(1.338)	(1.697)	(1.763)
Dioxin-only hit count is strictly positive (1:yes,0:no)	0.711	0.724	0.708	0.724	0.738	0.720						
	(0.416)	(0.389)	(0.384)	(0.423)	(0.395)	(0.389)						
Bombing load is strictly positive (1:yes,0:no)	5.871	4.623	4.933	5.977	4.704	5.029						
	(7.759)	(6.507)	(6.936)	(7.930)	(6.655)	(7.116)						
Logarithm of total bombing load	0.943	0.959	0.955	0.942	0.959	0.954	0.857	0.872	0.857	0.857	0.871	0.857
	(0.074)	(0.079)	(0.079)	(0.075)	(0.079)	(0.079)	(0.107)	(0.110)	(0.108)	(0.107)	(0.110)	(0.108)
Average daily sunshine over the period 1975-1999 (hours)	0.978	0.995	0.983	0.974	0.990	0.979	1.337	1.209	1.193	1.337	1.212	1.194
	(0.140)	(0.156)	(0.154)	(0.139)	(0.155)	(0.152)	(0.357)	(0.322)	(0.320)	(0.358)	(0.323)	(0.322)
Individual's gender (1:male,0:female)	0.602**	0.550***	0.651**	0.603**	0.550***	0.651**	0.415***	0.372***	0.442***	0.415***	0.372***	0.442***
	(0.130)	(0.110)	(0.126)	(0.130)	(0.109)	(0.127)	(0.114)	(0.107)	(0.125)	(0.114)	(0.107)	(0.125)
Individual's age (years)	1.160***	1.149***	1.149***	1.145***	1.137***	1.139***	1.230***	1.240***	1.241***	1.240***	1.270***	1.264***
	(0.039)	(0.044)	(0.044)	(0.046)	(0.051)	(0.047)	(0.061)	(0.064)	(0.066)	(0.079)	(0.076)	(0.076)
Individual's age squared	0.999***	0.999***	0.999***	0.999***	0.999**	0.999**	0.999***	0.998***	0.998***	0.998***	0.998***	0.998***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)
Individual lives in urban area (1:yes,0:no)	1.674***	1.676**	1.771***	1.677***	1.674**	1.771***	1.258	1.265	1.354	1.258	1.265	1.353
	(0.362)	(0.410)	(0.426)	(0.363)	(0.412)	(0.429)	(0.330)	(0.393)	(0.427)	(0.329)	(0.392)	(0.425)
Household size	0.999	1.003	1.022	0.999	1.003	1.023	1.010	1.036	1.054	1.010	1.037	1.054
	(0.043)	(0.043)	(0.044)	(0.043)	(0.043)	(0.044)	(0.045)	(0.040)	(0.039)	(0.045)	(0.040)	(0.039)
Individual's education (grade achieved)		1.104	1.066		1.109	1.073		0.935	0.910		0.938	0.912
		(0.142)	(0.137)		(0.145)	(0.140)		(0.144)	(0.138)		(0.142)	(0.137)
Logarithm of household per capita expenditure		0.896	0.905		0.896	0.906		1.133	1.152		1.128	1.148
		(0.129)	(0.136)		(0.130)	(0.137)		(0.167)	(0.168)		(0.166)	(0.168)
Individual seeked inpatient or outpatient care in past 12 months of survey (1:yes,(			5.257***			5.259***			4.663***			4.655***
			(0.963)			(0.962)			(0.797)			(0.794)
Constant	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Total effect of log dioxin-only hit count on post-1971 cohorts				*0.885*	*0.879*	*0.888*				*1.185*	*1.186*	*1.173*
				(0.207)	*(0.205)*	*(0.207)*				*(0.433)*	*(0.437)*	*(0.436)*
Number of observations	158,019	148,022	148,022	158,019	148,022	148,022	77,441	72,499	72,499	77,441	72,499	72,499

Notes: Standard errors in parenthesis, corrected for heteroskedasticity and province-level clustering. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent-level respectively. Observations are weighted using sampling weights. The conditional sample is restricted to observations with positive hit counts within 15km of commune center. The "Total effect of log dioxin-only hit count on post-1971 cohorts" in columns 4, 5, 6, 10, 11 and 12 is the product between the main effect ("Logarithm of dioxin-only hit count") and the interaction term ("Additional effect of log dioxin-only hit count on post-1971 cohorts"). The main effect then measures the effect of "log dioxin-only hit count" on pre-1971 cohorts.

Figure 1: Extending the buffer zone around commune administrative centers to measure herbicide exposure

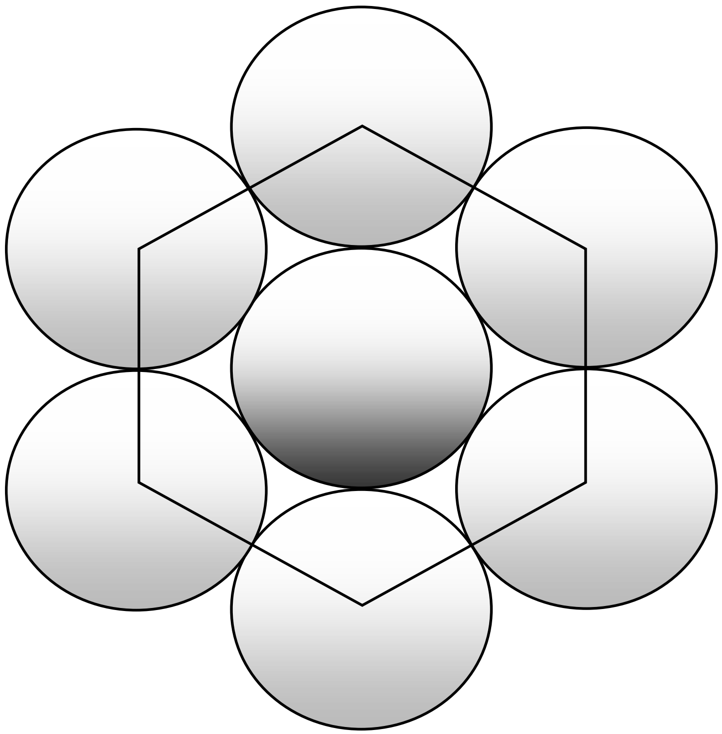

Figure 3: Correlation between military herbicide exposure and various measures of current expenditures

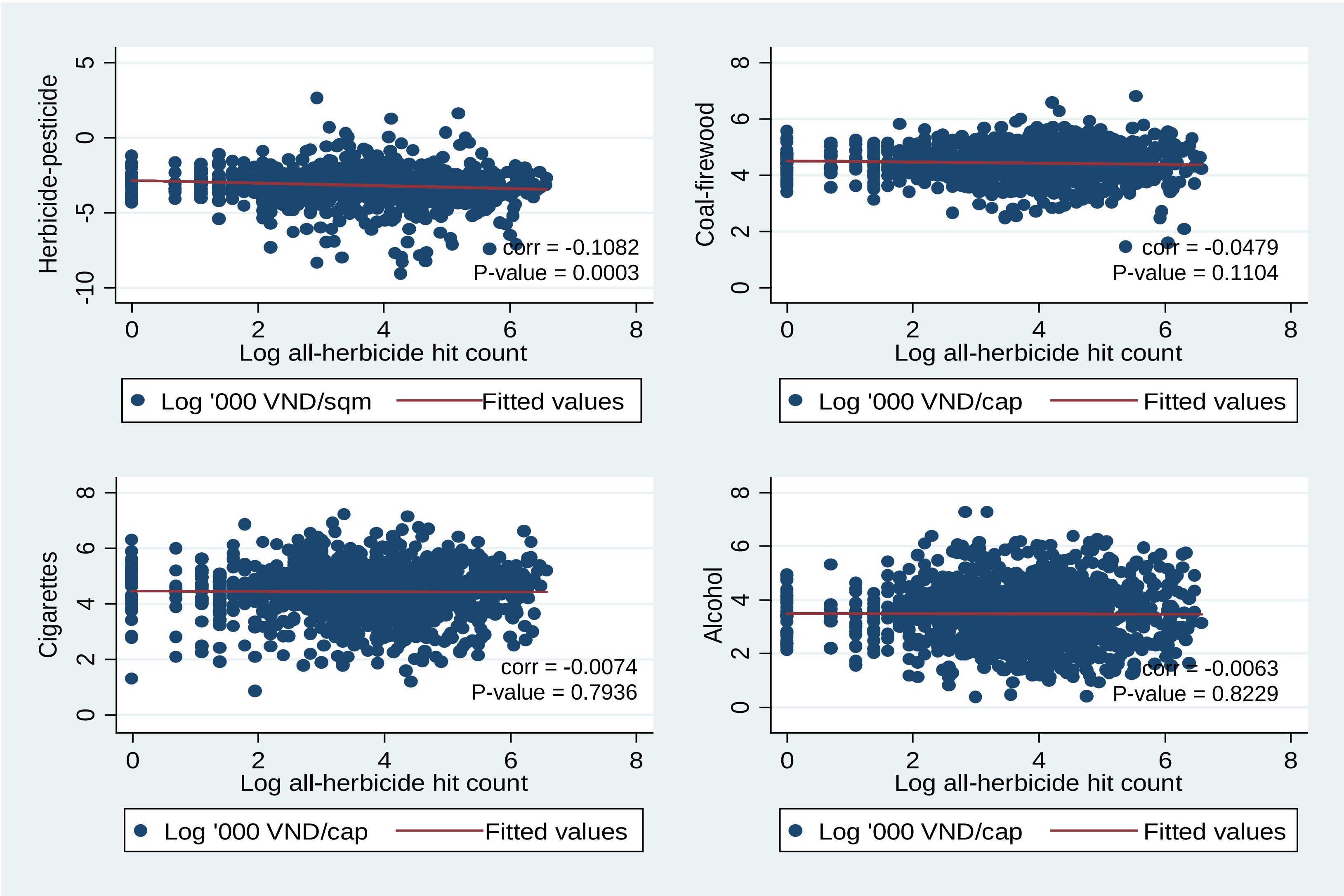

Table 1: Composition and quantities of herbicide mixtures sprayed 1961-1971

Name	Chemical constituents	Estimated quantities (liters)
Agent Pink	60%-40% n-Butyl: isobutyl ester of 2,4,5-T	50 312 sprayed - 413 852 on procurement records
Agent Green	n-Butyl ester 2,4,5-T	31 026 on procurement records
Agent Purple	50% n-Butyl ester 2,4,-D; 30% n-Butyl ester 2,4,5-T; 20% isobutyl ester 2,4,5-T	1 892 773
Agent Orange	50% n-Butyl ester 2,4,-D; 50% n-Butyl ester 2,4,5-T	45 677 937 (including Agent Orange II)
Agent Orange II	50% n-Butyl ester 2,4,-D; 50% isooctyl ester 2,4,5-T	at least 3 591 000 shipped
Agent White	Acid weight basis: 21.2%; tri-isopropanolamine salts of 2,4-D; 5.7% picloram	20 556 525
Agent Blue (powder)	Cacodylic acid and sodium cacodylate	25 650
Agent Blue (solution)	Cacodylic acid and sodium cacodylate	501 381

Source: Stellman et al. (2003)

Table 2: Summary statistics

	N. obs.	N. communes	mean	st. dev
Individual health outcomes (VNHS)				
Individual has cancer (1:yes,0:no)	158,019	1,200	0.08%	2.81%
Individual socio-economic characteristics (VNHS)				
Gender (1:male,0:female)	158,019	1,200	48.51%	49.98%
Age (years)	158,019	1,200	26.634	19.381
Education level (grade)	148,279	1,128	1.068	1.044
Household size	158,019	1,200	5.160	1.958
Per capita expenditure ('000 VND)	157,744	1,196	3,910	4,757
Live in urban area (1:yes,0:no)	158,019	1,200	22.80%	41.95%
Average daily sunshine hours over the period 1975-1999 (hours)	158,019	1,200	5.633	1.320
Individual seeked inpatient or outpatient care in past 12 months of survey (1:yes,0:no)	158,019	1,200	20.87%	40.64%
Commune-level characteristics (HERBS, iMMAP, and MDC)				
Commune showing positive number of herbicide hits within 15km		1,200	49.42%	50.02%
All-herbicide hit count within 15km		1,200	42.458	92.469
Commune showing positive number of dioxin hits within 15km		1,200	47.42%	49.95%
Dioxin-only hit count within 15km		1,200	19.388	42.260
Commune showing positive bombing load within 15km		1,200	77.58%	41.72%
Bombing load within 15km (M lbs)		1,200	38.146	120.577
Average daily sunshine over the period 1975-1999 (hours)		1,200	5.610	1.325
Secondary data: 2004 commune-level characteristics (VHLSS)				
Average agricultural herbicide and pesticide expenditure per square meter of agricultural land ('000 VND)		1,133	0.085	0.466
Average household per capita coal, firewood, sawdust, chaff ('000 VND)		1,114	98.269	62.429
Average household per capita alcohol and beer consumption ('000 VND)		1,246	58.572	92.486
Average household per capita cigarette consumption ('000 VND)		1,268	120.125	119.403

Remarks: Observations taken from VNHS are weighted using sampling weights.

Table 3: Logistic regression of the determinants of cancer - effect of all-herbice exposure

	Dependent variable: Individual reports suffering from cancer (1:yes,0:no)								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Independent variables									
All-herbicide hit count is strictly positive (1:yes,0:no)				0.815	0.792	0.813			
				(0.369)	(0.364)	(0.378)			
All-herbicide hit count within 15km above 75th percentile cutoff (1:yes,0:no)							1.228	1.166	1.201
							(0.284)	(0.279)	(0.290)
Bombing load is strictly positive (1:yes,0:no)	4.120	3.087	3.171	3.504	2.582	2.671	5.324	3.734	3.986
	(4.687)	(3.862)	(3.923)	(4.093)	(3.264)	(3.339)	(6.369)	(4.786)	(5.044)
Logarithm of total bombing load	0.964	0.983	0.981	0.974	0.995	0.992	0.948	0.972	0.967
	(0.064)	(0.071)	(0.071)	(0.066)	(0.072)	(0.071)	(0.067)	(0.073)	(0.072)
Average daily sunshine over the period 1975-1999 (hours)	0.984	1.011	0.999	1.048	1.086	1.064	0.956	0.989	0.974
	(0.090)	(0.095)	(0.092)	(0.176)	(0.199)	(0.195)	(0.088)	(0.095)	(0.092)
Individual's gender (1:male,0:female)	0.602**	0.550***	0.651**	0.602**	0.552***	0.652**	0.603**	0.550***	0.652**
	(0.130)	(0.110)	(0.127)	(0.129)	(0.110)	(0.127)	(0.130)	(0.110)	(0.127)
Individual's age (years)	1.160***	1.149***	1.149***	1.160***	1.149***	1.149***	1.160***	1.149***	1.149***
	(0.039)	(0.044)	(0.044)	(0.039)	(0.044)	(0.044)	(0.039)	(0.044)	(0.044)
Individual's age squared	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Individual lives in urban area (1:yes,0:no)	1.635***	1.655**	1.748***	1.660***	1.684**	1.776***	1.624**	1.650**	1.741***
	(0.328)	(0.391)	(0.406)	(0.343)	(0.405)	(0.419)	(0.332)	(0.393)	(0.409)
Household size	0.998	1.001	1.021	1.000	1.003	1.022	0.998	1.001	1.021
	(0.042)	(0.043)	(0.044)	(0.043)	(0.043)	(0.044)	(0.043)	(0.043)	(0.044)
Individual's education (grade achieved)		1.102	1.064		1.095	1.059		1.104	1.067
		(0.142)	(0.137)		(0.141)	(0.136)		(0.143)	(0.138)
Logarithm of household per capita expenditure		0.884	0.890		0.890	0.895		0.877	0.884
		(0.131)	(0.137)		(0.131)	(0.137)		(0.129)	(0.134)
Individual seeked inpatient or outpatient care in past 12 months (1:yes,0:no)			5.246***			5.240***			5.257***
			(0.963)			(0.962)			(0.965)
Constant	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Number of observations	158,019	148,022	148,022	158,019	148,022	148,022	158,019	148,022	148,022

Notes: Standard errors in parenthesis, corrected for heteroskedasticity and province-level clustering. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent-level respectively. Observations are weighted using sampling weights. The 75th percentile cutoff corresponds to 39 all-herbicide hits.

Table 4: Logistic regression of the determinants of cancer - dose response to all-herbicide hit counts

	Dependent variable: Individual reports suffering from cancer (1:yes,0:no)											
	Full sample						Conditional sample					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Independent variable												
Logarithm of all-herbicide hit count	1.198**	1.256**	1.270**	1.228**	1.291***	1.306***	1.238**	1.295**	1.319***	1.241**	1.307**	1.334**
	(0.104)	(0.126)	(0.126)	(0.110)	(0.134)	(0.134)	(0.117)	(0.137)	(0.138)	(0.123)	(0.149)	(0.150)
Additional effect of log all-herbicide hit count on post-1971 cohorts				0.779*	0.772*	0.771*				0.966	0.913	0.889
				(0.126)	(0.123)	(0.122)				(0.141)	(0.136)	(0.132)
Individual is born after 1971 (1:yes,0:no)				1.268	1.371	1.390				1.306	2.117	2.136
				(0.704)	(0.765)	(0.750)				(1.178)	(1.763)	(1.794)
All-herbicide hit count is strictly positive (1:yes,0:no)	0.421*	0.341**	0.337**	0.431*	0.352**	0.346**						
	(0.255)	(0.202)	(0.202)	(0.257)	(0.204)	(0.204)						
Bombing load is strictly positive (1:yes,0:no)	6.036	5.023	5.262	6.111	5.068	5.318						
	(7.479)	(6.639)	(6.876)	(7.597)	(6.724)	(6.987)						
Logarithm of total bombing load	0.942	0.955	0.951	0.942	0.955	0.952	0.878	0.868	0.852	0.878	0.868	0.852
	(0.069)	(0.073)	(0.073)	(0.069)	(0.073)	(0.073)	(0.104)	(0.102)	(0.100)	(0.104)	(0.103)	(0.100)
Average daily sunshine over the period 1975-1999 (hours)	1.042	1.079	1.057	1.038	1.074	1.054	1.335	1.216	1.201	1.336	1.218	1.202
	(0.168)	(0.190)	(0.186)	(0.167)	(0.188)	(0.185)	(0.341)	(0.314)	(0.314)	(0.341)	(0.316)	(0.315)
Individual's gender (1:male,0:female)	0.604**	0.553***	0.654**	0.605**	0.553***	0.654**	0.406***	0.365***	0.435***	0.406***	0.364***	0.435***
	(0.130)	(0.110)	(0.127)	(0.130)	(0.110)	(0.127)	(0.111)	(0.105)	(0.123)	(0.112)	(0.105)	(0.123)
Individual's age (years)	1.160***	1.149***	1.150***	1.146***	1.139***	1.141***	1.230***	1.243***	1.245***	1.240***	1.275***	1.269***
	(0.039)	(0.044)	(0.044)	(0.046)	(0.051)	(0.047)	(0.060)	(0.065)	(0.066)	(0.078)	(0.077)	(0.077)
Individual's age squared	0.999***	0.999***	0.999***	0.999***	0.999**	0.999**	0.999***	0.998***	0.998***	0.998***	0.998***	0.998***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)
Individual lives in urban area (1:yes,0:no)	1.620**	1.666**	1.754***	1.622**	1.662**	1.751***	1.265	1.237	1.316	1.265	1.238	1.316
	(0.345)	(0.407)	(0.421)	(0.347)	(0.409)	(0.423)	(0.332)	(0.372)	(0.399)	(0.332)	(0.373)	(0.399)
Household size	1.002	1.005	1.025	1.002	1.006	1.026	1.026	1.044	1.063	1.026	1.044	1.063
	(0.043)	(0.044)	(0.044)	(0.043)	(0.044)	(0.045)	(0.044)	(0.040)	(0.039)	(0.044)	(0.040)	(0.040)
Individual's education (grade achieved)		1.098	1.062		1.104	1.069		0.961	0.933		0.965	0.936
		(0.141)	(0.136)		(0.144)	(0.140)		(0.153)	(0.148)		(0.152)	(0.148)
Logarithm of household per capita expenditure		0.852	0.859		0.852	0.859		1.047	1.057		1.042	1.054
		(0.125)	(0.130)		(0.126)	(0.132)		(0.154)	(0.154)		(0.153)	(0.154)
Individual seeked inpatient or outpatient care in past 12 months of survey (1:yes,(			5.264***			5.269***			4.792***			4.788***
			(0.964)			(0.963)			(0.817)			(0.812)
Constant	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Total effect of log all-herbicide hit count on post-1971 cohorts				*0.957*	*0.996*	*1.007*				*1.200**	*1.193**	*1.185*
				(0.170)	*(0.183)*	*(0.182)*				*(0.169)*	*(0.163)*	*(0.164)*
Number of observations	158,019	148,022	148,022	158,019	148,022	148,022	81,268	75,983	75,983	81,268	75,983	75,983

Notes: Standard errors in parenthesis, corrected for heteroskedasticity and province-level clustering. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent-level respectively. Observations are weighted using sampling weights. The conditional sample is restricted to observations with positive hit counts within 15km of commune center. The "Total effect of log all-herbicide hit count on post-1971 cohorts" in columns 4, 5, 6, 10, 11 and 12 is the product between the main effect ("Logarithm of all-herbicide hit count") and the interaction term ("Additional effect of log all-herbicide hit count on post-1971 cohorts"). The main effect then measures the effect of "log all-herbicide hit count" on pre-1971 cohorts.

Table A1: Logistic regression of the determinants of cancer - effect of dioxin-only exposure

	Dependent variable: Individual reports suffering from cancer (1:yes,0:no)								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Independent variables									
Dioxin-only hit count is strictly positive (1:yes,0:no)				1.023	1.053	1.057			
				(0.412)	(0.424)	(0.427)			
Dioxin-only hit count within 15km above 75th percentile cutoff (1:yes,0:no)							1.421	1.367	1.375
							(0.364)	(0.383)	(0.394)
Bombing load is strictly positive (1:yes,0:no)	4.120	3.087	3.171	4.214	3.242	3.348	6.466*	4.677	4.857
	(4.687)	(3.862)	(3.923)	(5.030)	(4.104)	(4.192)	(8.249)	(6.491)	(6.696)
Logarithm of total bombing load	0.964	0.983	0.981	0.962	0.980	0.978	0.937	0.958	0.955
	(0.064)	(0.071)	(0.071)	(0.068)	(0.072)	(0.071)	(0.071)	(0.078)	(0.078)
Average daily sunshine over the period 1975-1999 (hours)	0.984	1.011	0.999	0.978	0.996	0.983	0.936	0.966	0.955
	(0.090)	(0.095)	(0.092)	(0.142)	(0.159)	(0.156)	(0.089)	(0.096)	(0.093)
Individual's gender (1:male,0:female)	0.602**	0.550***	0.651**	0.602**	0.550***	0.651**	0.603**	0.550***	0.651**
	(0.130)	(0.110)	(0.127)	(0.129)	(0.110)	(0.127)	(0.130)	(0.110)	(0.126)
Individual's age (years)	1.160***	1.149***	1.149***	1.160***	1.149***	1.149***	1.160***	1.149***	1.149***
	(0.039)	(0.044)	(0.044)	(0.039)	(0.044)	(0.044)	(0.039)	(0.044)	(0.044)
Individual's age squared	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***	0.999***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Individual lives in urban area (1:yes,0:no)	1.635***	1.655**	1.748***	1.632**	1.647**	1.740***	1.650***	1.649**	1.742***
	(0.328)	(0.391)	(0.406)	(0.345)	(0.399)	(0.413)	(0.342)	(0.397)	(0.412)
Household size	0.998	1.001	1.021	0.997	1.001	1.020	0.998	1.002	1.021
	(0.042)	(0.043)	(0.044)	(0.043)	(0.043)	(0.043)	(0.043)	(0.043)	(0.044)
Individual's education (grade achieved)		1.102	1.064		1.103	1.065		1.107	1.070
		(0.142)	(0.137)		(0.142)	(0.136)		(0.143)	(0.137)
Logarithm of household per capita expenditure		0.884	0.890		0.882	0.888		0.898	0.906
		(0.131)	(0.137)		(0.128)	(0.134)		(0.136)	(0.142)
Individual seeked inpatient or outpatient care in past 12 months of survey (1:yes			5.246***			5.246***			5.249***
			(0.963)			(0.963)			(0.963)
Constant	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Number of observations	158,019	148,022	148,022	158,019	148,022	148,022	158,019	148,022	148,022

Notes: Standard errors in parenthesis, corrected for heteroskedasticity and province-level clustering. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent-level respectively. Observations are weighted using sampling weights. The 75th percentile cutoff corresponds to 19 dioxin-only hits.